Amendments to the Specification:

**Please replace the paragraph number [00015] (at page 6) with the following amended paragraph [00015]:**

**[00015]** Diabetic autoantigen-specific T cells (and autoantigen-specific T cells in general) are responsible for the proliferation of the immune response to the autoantigen which they display. The immune response results in inflammation and eventual destruction of β cells of the pancreatic islets and the development of frank symptoms of diabetes. However, some antigen-specific T cells have been difficult to detect and study due to their low numbers and to the generally low affinity with which they bind to their antigen. New methods to detect the presence of these antigen-specific T cells, particularly methods which are able to detect infrequent cells or cells which bind with low affinity, are needed. These methods would be able to detect a pre-diabetic state or other pre-autoimmune disease state before overt symptoms of the disease develop. Reagents which are able to detect members of specific T cell clones also may be used to provide methods of modulating the activity of the T cell clones, for example, inducing tolerance, expanding the clone or killing undesired T cells which bind the specific antigen. In addition, specific T cell clone identification can be useful for studying the effect of antigen treatment of IDDM by isolating the T ~~cells~~ cell clones produced in response to the treatment.

**Please replace paragraph number [00046] (at page 14) with the following amended paragraph [00046]:**

**[00046]** Figure 17 shows fluorescence activated cell sorting results characterizing purified peptide-specific T cells from NOD (Figures A-D) or BALB/c (Figures E-H) mice by staining with I-Ag7 or I-Ad ~~tetremer~~ tetramer reagents and an anti-CD4 antibody.

**Please replace paragraph number [00087] (at page 31) with the following amended paragraph [00087]:**

**[00087]** The inhibitory effect of tetramer+ T cells could be due to differential expansion or homeostatic competition of tetramer+ T cells vs. diabetogenic splenocytes in animals as described in Theofilopoulous et al., J. Clin. Invest. 108:335-340, 2001; Salomon et al., Annu. Rev. Immunol. 19:225-252, 2001. The tetramer+ T cells ~~hay~~ may have a growth advantage over the diabetogenic splenocytes. The low numbers of N206+ or N221+ cells (2 to 4%) detected in the spleens of the co-transferred animals suggest that they proliferated slowly but were sufficient to inhibit diabetes.

**Please replace paragraph number [000120] (at page 48) with the following amended paragraph [000120]:**

**[000120]** Cultured splenocytes from either NOD mice Figures 17A-D or BALB/c mice Figures 17E-H which had been immunized with the indicated peptides were isolated with the corresponding tetramer and stained with anti-CD4 and labeled tetramers as indicated.

The results shown in Figure 17 were typical of at least four different experiments. Consistent with previous studies, NOD mice immunized with IFA alone, unlike those immunized with CFA, could still develop diabetes (data not shown). Therefore, IFA treatment did not itself alter the function of T cells critical to diabetes development. The I-Ag7 and I-Ad tetramers were shown to be ~~antigen specific~~ antigen-specific as they did not detect a significant number of T cells for mice immunized with the other peptide.